1: Biochem Biophys Res Commun 1997 Nov 26;240(3):580-5 Related Articles, Links

ELSEVIER SCIENCE FULL-TEXT ARTICLE

The rat S-adenosylhomocysteine hydrolase promoter.

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The 1.2-kb DNA sequence flanking the transcription start of the AdoHcy hydrolase gene was cloned into the luciferase reporter plasmid pGL3-basic, and promoter activity was measured in transiently transfected CHO cells. Deletion analysis showed that most promoter activity was located within a 153 bp fragment immediately upstream from the predominant transcription start. The 153 bp fragment includes sites for AP-2, glucocorticoid-responsive element, SP-1, and a TATA-like sequence TATTTAAA. Mutational analysis demonstrated that the SP-1 site nearest the start of transcription contributed significantly to promoter activity, whereas, the other elements, including the appropriately positioned TATTTAAA sequence, had little affect on promoter activity.

PMID: 9398607 [PubMed - indexed for MEDLINE]

2: Eur J Biochem 1995 Apr 15;229(2):575-82 Related Articles, Links

The gene and pseudogenes of rat S-adenosyl-L-homocysteine hydrolase.

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Two rat liver genomic DNA libraries constructed in lambda DASH and lambda Charon 4A were screened for sequences with similarity to S-adenosyl-L-homocysteine (AdoHcy) hydrolase cDNA. Of 36 clones purified, two contained the AdoHcy hydrolase gene sequence and 34 contained pseudogene sequences. The AdoHcy hydrolase gene, which has been sequenced in its entirety, spans approximately 15 kb and consists of 10 exons. Primer extension and S1 experiments show that transcription is initiated from two major initiation sites located at positions -63 and -62 from the starting codon and from several minor sites. The promoter region is located in a CpG island, sequence TATTTAAA is present 23 bases upstream from the transcription start site, and an inverted CCAAT box is located 285 bp upstream from the transcription start site. Other potential transcription-factor binding sites including SP1, AP-2, GRE and Oct-1 sites were identified in the 5'-flanking region. Several different processed pseudogenes were found and analyzed.

PMID: 7744082 [PubMed - indexed for MEDLINE]

3: J Biol Chem 1994 Feb 11;269(6):4084-91 Related Articles, Links

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The role of cysteine 78 in fluorosulfonylbenzoyladenosine inactivation of rat liver S-adenosylhomocysteine hydrolase.

Aksamit RR, Backlund PS Jr, Moos M Jr, Caryk T, Gomi T, Ogawa H, Fujioka M, Cantoni GL.

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Inactivation of rat liver S-adenosylhomocysteine hydrolase by the site-directed reagent 5'-p-fluorosulfonylbenzoyladenosine (FSBA) is associated with the formation of a disulfide bond between Cys-78 and Cys-112 (Takata, Y., and Fujioka, M. (1984) Biochemistry 23, 4357-4362; Gomi, T., Ogawa, H., and Fujioka, M. (1986) J. Biol. Chem. 261, 13422-13425). To characterize the inactivation mechanism more precisely, the properties of four hydrolase proteins mutated at Cys-78 or Cys-112 were compared to those of the wild-type enzyme. When Cys-78 was mutated to either a serine or an alanine, proteins with greatly reduced enzymatic activity were obtained, large effects on kinetic constants were observed, and enzymatic activity was not affected by incubation with FSBA. When Cys-112 was mutated to either a serine or an alanine, the activity was similar to the wild-type protein, only small changes in the kinetic constants were observed, and the enzyme was inactivated more rapidly upon incubation with FSBA. FSBA inactivation of the C112A mutant protein was accompanied by the formation of a disulfide between Cys-78 and Cys-52. The data indicate that FSBA initially reacts with Cys-78 and that Cys-78 has an important role in the structure of the enzyme.

PMID: 8307967 [PubMed - indexed for MEDLINE]
